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LETTER TO THE EDITOR

Role of serum quantitative hepatitis B surface antigen (HBsAg) in HBsAg carriers in a follow-up community study

To the Editor,

Readers raised valuable comments to our manuscript entitled "Changing serum levels of quantitative hepatitis B surface antigen (HBsAg) and hepatitis B virus DNA in hepatitis B virus surface antigen carriers: A follow-up study of an elderly cohort", which was published in the *Kaohsiung Journal of Medical Sciences* [1]. This is a community-based study in a hepatitis B, C, and D virus (HBV, HCV, and HDV, respectively) endemic area. In the initial screening in 1997, 2909 (30.2% of the whole population in the township) adults aged $\geq$ 45 years [2,3] were enrolled. Among them, only 222 (7.6%) were hepatitis B surface antigen-positive [HBsAg(+)], anti-HCV(−), and anti-HDV(−). Thirteen years later (2010), 59 of these 222 individuals (26.6%) responded to this follow-up study. The small sample size is one of the limitations of this study. It should be noted that sample size depends on the prevalence of diseases and the response rate of study participants. It is also more difficult to conduct such a long-term follow-up study for older populations. However, this type of study provided better community-based information compared with a well-known hospital-based study. In a small part of our previous study in this population [3], 23 of 25 (92%) individuals were genotype B and only two (8%) were genotype C. Using this proportion, only about five individuals were genotype C among these 59 chronic hepatitis B patients. Having only five cases should not result in any conclusion. For cost—benefit consideration, we did not include the expensive genotype test in this study. Meanwhile, the contribution of genotype in HBsAg is still controversial. Although the secretion of HBsAg might be strongly influenced by HBV genotype [4], the previous study also indicated that HBV genotype B or C was not associated with HBsAg clearance in HBeAg-negative patients [5]. As mentioned above, this was a community-based study, and the study participants had only a mild liver disease. In our study cohort, all 59 patients were diagnosed by ultrasound to have parenchymal liver disease or fatty liver disease in 2005. Only two cases progressed to liver cirrhosis in 2010; both were in the high HBsAg and high HBV DNA (HsHD) groups persistently. Moreover, none of the patients developed hepatocellular carcinoma (HCC) during follow-up. Therefore, advanced liver diseases such as cirrhosis or HCC were not main the concern of the HBsAg decrease in our study.

We emphasized that this community-based study provided clinical information from asymptomatic residents.

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Conflicts of interest: All authors declare no conflicts of interest.

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